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The following paper was read :

NERVOUS AFFECTIONS FOLLOWING INJURY, "CONCUSSION OF THE SPINE," "RAIL- WAY SPINE" AND "RAILWAY BRAIN."

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It is rather singular that the two most elaborate works on those affections of the nervous system which are supposed to follow injury should have been written by surgeons, and should have been based on the evidence of railway cases. One of these works, the influence of which is not yet dead, is based upon the ideas in regard to the pathology of the nervous system which obtained twenty years ago, and treats of all forms of injury of the central nervous system under the most misleading heading of "concussion of the spine;" the other, which reads like the work of a special pleader for the railway companies, discusses case after case of obscure nervous disease without mention of the condition of the reflexes. Fortunately, however, the attention of neurologists has of late been directed to the subject, and since the appearance of Page's first treatise in 1881, many valuable contributions to our knowledge have been made in this country and in Europe, and the work done in Germany, especially, has brought the matter more fully to our attention.

Erichsen's composite of "concussion of the spine" has been found to be compounded of too many distinct conditions to be trustworthy, and, from the vagueness of his classification, his ideas on prognosis proved misleading; yet his work has had such an influence that the English railway companies are said to have paid eleven million dollars in damages in five years, and I have no question that it has also had an influence upon the great sums that

have been paid in this country. The reaction, of course, followed, and it was aided by the cynicism that naturally arises when we see a man, who has claimed to be permanently injured, walk off as well as ever when the "damages" have been paid. It has seemed to me, however, that this reaction has gone too far, and therefore I have thought it worth while to go over the subject once more, and to review some of the recent work that has been done upon it. Before discussing controverted points, however, I will mention briefly certain definite lesions of the nervous system which may unquestionably follow injury.

Among the commoner results of injury are the affections of the peripheral nerves. The obstinate pain and persistent weakness of the shoulder which so often follow an injury are probably due to an implication of certain nerve fibres in the periarthritic process. Beside that, we often see various local paralyses, due to all sorts of lesions of the nerves, from simple pressure to severe crushing,—paralyses of all forms of intensity, from the transitory forms with normal electrical reactions to the severe atrophic forms with reaction of degeneration. The prognosis, of course, varies with the degree of injury, and is governed by the ordinary rules.

Trauma may produce certain definite lesions of the spinal cord and its coverings, beside the vague and questionable results of pure "concussion." It may, in the first place, cause fracture or dislocation of the vertebræ, and, secondarily, affect the cord itself. In these cases it not infrequently happens that the patient exhibits the symptoms of injury to the cord, while the injury to the vertebræ is noted only at the autopsy. With or without injury of the vertebræ, however, we may find serious injury to the cord,—hæmorrhage into the meninges or into the cord itself, rupture of the pia with hernia of the cord, or acute myelitis.¹

In addition to the cases of what may be called "acute injury to the cord," where the symptoms develop immediately after the accident, it is a well-attested fact that

¹ E. Leyden. *Klinik der Rückenmarkskrankheiten*, i. 371, ii. 61, 92, 139.

chronic degenerative processes may be due to injury; and here, of course, the symptoms are very insidious in their onset. Spitzka² and Gowers³ cite cases of tabes dorsalis due to injury, and Hoffmann⁴ has just reported a very interesting case of tabes from Erb's clinique at Heidelberg due to a prolonged daily concussion of the whole body, especially the abdomen. Dana,⁵ too, has cited a case where tabetic symptoms followed a railway injury to a syphilitic subject, where he thinks the accident determined the localization of the morbid process. Besides tabes, injury may produce lateral sclerosis, progressive muscular atrophy,⁶ diffuse sclerosis,⁷ and disseminated sclerosis,⁸—the last two affections especially being extremely difficult to diagnosticate in their early stages.

There is, or rather was, another lesion of the cord which was once deemed of great importance and was regarded by Erichsen⁹ as the chief source of the symptoms of his "concussion of the spine,"—namely, spinal lepto-meningitis. We used to hear of it, but lately the cases have become rare, and, in fact, few now disagree with Strümpell's dictum,¹⁰—"A case of primary chronic lepto-meningitis, which can be surely and convincingly proven clinically and anatomically, does not exist."

Finally, in the brain and its coverings injury may produce various lesions,—fracture of the skull, meningeal and intra-cerebral hæmorrhage, pachymeningitis interna, hæmorrhagica, meningitis, softening, abscess, tumor, and various functional disorders, such as epilepsy, paralysis agitans,

² E. C. Spitzka. The Chronic Inflammatory and Degenerative Affections of the Spinal Cord. *Pepper's System of Medicine*, v. 855.

³ W. R. Gowers. *Diseases of the Nervous System*, i. 289.

⁴ J. Hoffmann. Beitrag zur Ätiologie, Symptomatologie und Therapie der Tabes dorsalis. *Archiv f. Psychiatrie u. Nervenkrankheiten*, xix. 438, 1888.

⁵ L. Dana. Nervous Syphilis following a Railroad Injury. *The Post-Graduate*, April, 1888.

⁶ W. R. Gowers. *Op. cit.*, i. 450.

⁷ W. R. Gowers. *Op. cit.*, i. 238.

⁸ E. C. Spitzka. *Op. cit.*, p. 884.

⁹ J. E. Erichsen. On Concussion of the Spine, p. 85.

¹⁰ A. Strümpell. *Lehrbuch der speciellen Pathologie und Therapie der inneren Krankheiten*, ii. 1, 450, 4te Aufl.

and chorea.¹¹ Furthermore, injury may give rise to various psychoses¹² and chronic degenerative processes, especially paretic dementia,¹³ and of some of these, and of certain functional nervous affections, I will speak later.

Thus far all is clear and well defined. It would require an exhaustive treatise to speak fully of all these conditions and to dwell on their diagnosis and prognosis. They are met with more or less often, and usually they can be readily recognized. Besides these affections, however, there are other cases of a more obscure character, where our diagnosis is often doubtful and our prognosis sadly at fault.

Whether there is a true "concussion of the spinal cord" is still a matter of doubt. By this term I mean a paraplegia following injury, where the cord has sustained no coarse mechanical lesion, where "molecular changes in the finer nerve-elements have occurred, giving rise to an immediate and complete functional paralysis,"¹⁴—a condition analogous to the commoner concussion of the brain. Page¹⁵ questions the possibility of such an affection, but cases have been reported which clinically answer the requirements.¹⁶ The anatomical relations of the cord naturally render it difficult for true concussion to occur; and, moreover, in simple concussion there is apt to be recovery, so that post-mortem evidence is lacking. Cases have been reported,¹⁷ however, where paraplegia came on suddenly after injury and terminated fatally, although no lesion could be found after death. Some of these cases are, of course, untrustworthy, as they were observed at a time when the methods of examining the cord were less exact, so that it is hard to exclude the existence of contusion or punctate hæmorrhages into the

¹¹ Ch. Bataille. *Traumatisme et Névropathie*.

¹² Hartmann. *Ueber Geistesstörungen nach Kopfverletzungen*. *Archiv f. Psychiatrie u. Nervenkrankheiten*, xv, 98, 1884.

¹³ R. v. Krafft-Ebing. *Lehrbuch der Psychiatrie*. i. 166.

¹⁴ W. H. Erb. *Diseases of the Spinal Cord*. *Ziemssen's Cyclopædia*, xiii. 347.

¹⁵ H. W. Page. *Injuries of the Spine and Spinal Cord*, p. 33.]

¹⁶ Wm. Hunt. *Concussion of the Brain and Spinal Cord*. *Pepper's System of Medicine*, v. 913.

¹⁷ E. Leyden. *Op. cit.*, ii. 93.

cord. Duménil and Petel,¹⁸ however, still hold to a belief in commotion of the cord, which may be the origin of consecutive inflammatory lesions or sclerosis, and Dana¹⁹ admits the existence, rarely, of true concussion. Some writers, Obersteiner²⁰ among them, hint at the existence of chronic concussion in men who are constantly exposed to jarring, as railway employes, but such cases are more likely to be classed among the degenerations of the cord, as in Hoffmann's case already cited.

Beside the true concussion there are a host of obscure affections which have been classed by Erichsen under the general head of "spinal concussion," and about which there has been much controversy. Dr. R. M. Hodges,²¹ in a paper read before the Boston Society for Medical Improvement eight years ago, was one of the early dissenters from the views of Erichsen. He showed that a strain of the muscles or ligaments of the spine was capable of explaining many of the symptoms, and he believed that many of the cases were cases of functional nervous disease. Soon after P'age²² advocated the same views with somewhat more detail, ascribing the symptoms in cases of "railway spine" to a traumatic lumbago (that is, a strain) and a traumatic neurasthenia caused by the shock and terror of the accident.

Two years later Walton²³ found that in a number of cases of injury there was anæsthesia or hemianæsthesia, often involving the special senses, and, calling our attention²⁴ anew

¹⁸ Duménil and Petel. Commotion de la moelle épinière. Archives de Neurologie, Jan., Mar., May, 1885.

¹⁹ C. L. Dana. Concussion of the Spine and its Relation to Neurasthenia and Hysteria. Medical Record, 6th Dec., 1884.

²⁰ H. Obersteiner. Ueber Erschütterung des Rückenmarks. Med. Jahrbücher, p. 531, 1879.

²¹ R. M. Hodges. So-called Concussion of the Spinal Cord. Boston Medical and Surgical Journal, 21st, 28th April, 1881.

²² H. W. Page. *Op. cit.*, p. 116 *et. seq.* Also Boylston Prize Dissertation for 1881.

²³ G. L. Walton. Two Cases of Hysteria. Archives of Medicine, Aug., 1883.

²⁴ G. L. Walton. Possible Cerebral Origin of the Symptoms usually classed under "Railway Spine." Boston Medical and Surgical Journal, 11th Oct., 1883.

to the fact that many of the symptoms were cerebral, he suggested the term "railway brain," as more suitable than "railway spine." About the same time Putnam²⁵ reported similar cases, and they both suggested the relation between hemianæsthesia and hysteria. This theory has been further elaborated by Charcot,²⁶ who states that "these grave and obstinate nervous states, which are presented as the result of railway collisions, rendering their victims unable to return to their work or resume their ordinary occupations for periods of several months or even years, are often only hysteria, nothing but hysteria." Much of Charcot's last volume is devoted to the description of cases of traumatic hysteria, and a number of his pupils have published further studies upon the subject.

Before all this, in 1880, Westphal²⁷ had reported three cases of "railway spine," and had advanced the theory that the symptoms were due to small foci of myelitis or encephalitis caused by trauma, and that they were analagous in their symptoms to multiple sclerosis. Since then Westphal's assistants, Thomsen and Oppenheim, have made²⁸ an elaborate study of sensory disturbances in all forms of nervous disease, including railway spine, and have shown that hemianæsthesia is not pathognomic of hysteria; and Oppenheim,²⁹ in a later paper, has carefully studied a second series of cases of "railway spine," with the result of substantiating Westphal's views.

²⁵ J. J. Putnam. Recent Investigations into the Pathology of so-called Concussion of the Spine, etc. Boston Medical and Surgical Journal, 6th Sept., 1883. The Medico-legal Significance of Hemianæsthesia after Concussion Accidents. American Journal of Neurology and Psychiatry, Nov., 1884.

²⁶ J. M. Charcot. Leçons sur les maladies du système nerveux, iii, 251.

²⁷ C. Westphal. Einige Fälle von Erkrankung des Nervensystems nach Verletzung auf Eisenbahnen. Charité-Annalen, v, 379, 1878.

²⁸ R. Thomsen und H. Oppenheim. Ueber das Vorkommen und die Bedeutung der sensorischen Anästhesie bei Erkrankungen des centralen Nervensystems. Archiv f. Psychiatrie u. Nervenkrankheiten, xv, 559, 633. 1884.

²⁹ H. Oppenheim. Weitere Mittheilungen über die sich am Kopfverletzungen und Erschütterungen (in specie: Eisenbahnunfälle) anschliessenden Erkrankungen des Nervensystems. Archiv f. Psychiatrie u. Nervenkrankheiten, xvi, 743. 1885.

Before discussing these various theories I will cite, as briefly as possible, some cases of nervous affections following injury that I have seen in the last three years. I have not selected these cases in support of any theory, but I have picked out cases of different types, representing as fairly as possible the whole number of cases that I have seen. In only three cases was there any question of damages. One of these was a child, and another was seen after the award had been made, although a question of appeal was pending. Thus we can eliminate at the start two factors which have tended to obscure the subject and to bias opinion—the idea of simulation and the excitement that naturally attends litigation and is often a hindrance to recovery. This gives a more satisfactory basis to reason from, for, as the late Dr. Curtis said³⁰ in the discussion of Dr. Hodges' paper, "treatises based, like Erichsen's work"—and the same may be said of Page and Rigler—"upon the evidence of railway cases are certainly the last sources of information from which one may learn to make a correct diagnosis and prognosis, and to escape being deceived by the voluntary or involuntary exaggeration and simulation so commonly observed in plaintiffs seeking damages." The sub-heading of "railway" spine and brain is hardly appropriate, for railway accidents were not the cause of the trouble in the majority of cases, and none of them were victims of a great railway accident, like that at Roslindale. Of course a railway accident has no specific effect, except that in it are brought to play the most tremendous forces that we employ in our daily lives, and the terror and horror of any great railway catastrophe has a vastly greater psychological effect.

I give the cases as briefly as possible, omitting unessential symptoms. I regret that in some of them my investigations in the domain of the special senses have not been as thorough as I could have wished.

I. Jeremiah C., 37, m., railway employee, consulted me in March, 1887. A year and a half before he was knocked

³⁰ Boston Medical and Surgical Journal, 5th Feb., 1880.

off a cable car, striking his back, and losing consciousness. On return of consciousness he went back to work, and kept at it for an hour or two, but afterwards he was laid up for seven weeks. Now he has pain in the back, especially on motion, with rigidity of the spine, and lumbar tenderness. His arms feel helpless; he has numbness and tingling in the hands and at times in the legs; the legs are not as strong, and he has had cramps in them. Occasional vertigo, and rush of blood to the head. Nervous, fretful, low-spirited, and poor memory. Some vesical tenesmus, and loss of sexual power. Some exaggeration of knee-jerks. Some improvement under faradism and the actual cautery.

II. John D., 30, m., organ finisher, consulted me in February, 1888. Fell down stairs a month ago, striking small of back and buttocks. Great pain in the back. Diminished power in left leg. He cannot bend his spine, and has great tenderness in the lumbar region. He has a desire to empty his bladder most of the time, and, when he passes water, he thinks he is through before he really is, occasionally wetting himself. No sexual power since the accident. Quite nervous and rather alarmed as to his condition. Knee-jerks rather quickened, a tap setting up a general shrinking, as if from pain.

III. Martin H., 46, m., draw-tender. Referred to me at the Boston Dispensary, in August, 1886. Two years ago fell from a mast, thirty-six feet, striking back. Since then has had sharp pains in the back and abdomen, shooting down the legs. The legs are easily fatigued, feel numb and prickly, and as if a pad were between them and the floor. "Drawing" girdle sensation. Twisting or bending the spine, or riding in the cars, is painful. Faint spells and vertigo; severe headache at times. Nervous, low-spirited, and a poor sleeper. Short breath, palpitation, and a "drawing" feeling in the stomach. Poor appetite and digestion. Arms feel numb and fingers feel as if asleep. Diminished sensation in arms and legs, and some tenderness of nerve-trunks in legs. Lumbar spine flat, slight lateral curvature to the right, tender below tenth dorsal vertebra, the tenderness being greater by the sides of the

spinous processes. Reflexes and electrical reactions normal. No ataxia.

IV. Bateman C., 59, m., electrician. January, 1886. Some months before he fell from a ladder, striking on his buttocks. No loss of consciousness. Nausea and vomiting till two months ago. Costive. Since accident loss of power and prickly throbbing in legs, worse in the right leg, which has wasted. Legs at times feel hot or cold, both subjectively and objectively. No distinct pain in legs. Water had to be drawn for a week after his fall. A month ago fell, rupturing a vessel in his knee, and the knee had to be aspirated. Right leg two inches smaller, marked diminution of sensibility. Muscles of thigh do not react to either current on right, and very feebly to strong galvanic current on left. Much fibrillary contraction of right quadriceps. Distinct gain under galvanism. With improvement in strength and sensation in legs has decided pain in them.

V. Jeremiah O'D., 50, m., carpenter. Consulted me in August, 1885, being anxious to get a pension on account of his disability. Was stabbed in the abdomen in 1865, and had peritonitis after it. In 1870 the pension board rejected his application, thinking his hemiparesis was the result of apoplexy, but he denies any history of apoplexy. On recovery from the peritonitis, the left leg began to be weak, and he had pain and stiffness in the left hip. He could not walk without staggering and getting exhausted. Hard to lift left leg up stairs. Severe pain in left side and abdomen, and left side of head. Depressed, poor memory, slight mental impairment, vertigo, and diplopia; some tinnitus. Numbness gradually developed over his entire left side, less marked in the hands and feet, but amounting in some places to absolute anæsthesia and analgesia. Tingling and prickling on left side. Smell impaired; poor vision in left eye from cataract, field not contracted; taste poor on left; hearing worse on left. Diminished tactile sensibility over entire left side; left arm a little smaller and weaker. Cannot put left leg into a chair without great effort. Sways with eyes shut. Knee-jerk and cremaster reflex most marked on right; knee-jerk weak. Speech rather indis-

tinct. Slight tenderness over left posterior tibial. March, 1888: Question of pension still pending. Has not improved since 1885. Symptoms much the same. Still has anæsthesia, which is most marked on the left, although tactile sensibility is blunted on the right. Considerable difficulty in walking, drags left foot. Trouble in locomotion increased on trying to make any quick movement.

VI. Susan W., 46, m. December, 1886. Neurotic taint. Fell on ice last winter, striking left hip and elbow, and causing hernia. Now the slightest effort causes pain across the chest and in the back. Lifting causes a "hot water" feeling in the hernia. Very severe headache, impaired vision, and increase of deafness in left ear. Tinnitus. Short breath and pleuritic stitch. Weak stomach; very costive; frequent micturition. Considerable pain in the arms, numb feeling on left side; the left hand and foot get cold readily. Staggers on walking, and the left leg gives out. Cramps in the legs; numbness, prickling, and pain in the left leg. Great spinal tenderness; tender over stomach and lower ribs on each side. Tender over left ulnar, sciatic, and posterior tibial nerves. Diminished sensibility in left ulnar region, over left chest, and on outer side of left leg. Electrical reactions normal. Knee-jerks exaggerated, front-tap contraction. Eyes and ears not examined. March, 1888: Worse since last seen. Pain in left side and back; prickly feeling all over body. Much vertigo. Pain in right foot. Poor vision. Very nervous. At times has much trouble in passing water. Troubled greatly with leucorrhœa and piles. Field of vision good, *von* 20-50. Fundus normal. Cannot hear watch with either ear, or through bone. Marked opacity of membrana tympani. Cannot stand with eyes shut. Slight tremor of hands. Extreme spinal tenderness.

VII. Annie S., 45, m. Seen in consultation February, 1886, with Dr. E. S. Boland, who has reported the case in full.³¹ Not neurotic. Two years before she was thrown down an embankment by the sudden starting of a train, and had recovered damages, although the case was still in dis-

³¹ E. S. Boland. Symptoms following Injury to the Head and Back. Boston Med and Surg. Journal, 10th Nov., 1887.

pute. Much pain in head and lumbar region, and considerable vertigo. Sleeps poorly and has a poor memory. Poor appetite; very costive; has had jaundice since the accident. At one time had xanthopsia, and another melanopsia. Menses irregular and painful. Urine scanty. Wets and soils herself at times. Very marked anæsthesia over whole body, with analgesia. Some sensation in tip of nose, left ulnar region, and right cheek. All muscular efforts slow and weak. Cannot stand without support. Muscles do not react well to faradism; knee-jerks weak. Field of vision contracted, especially in right eye; *rod*, can count fingers; *ros*, 2-20. Monocular diplopia, *od*. Pupils react sluggishly to light. Loss of smell and taste. Hearing to watch, contact *ad*, four inches, *as*. Gained under treatment for three months. Later right ankle became weak. When last heard from, December, 1887, she was still far from well, being quite lame, and having much pain in her back.

VIII. Chas. L., 14, s., school-boy. Referred to me by Dr. Cutter, of Leominster, in May, 1886. Nervous heredity. Posthumous child, always nervous and irritable, had convulsions in infancy. Six years ago fell from a bridge, striking forehead. Signs of shock after it. "Shoulders drew up and spine got crooked." Delirious after fall, and very nervous since. Said to have lateral curvature, but it was not detected. Three years ago eyes began to trouble him, with dim vision and pain. Much headache, irritable, surly, and heedless. Poor appetite, chronic diarrhœa. Palpitation. Passes much urine. Rheumatic fever a year ago. Muscles weak, pain in legs with numbness and prickling in hands and arms. Fell again last fall, striking head. Worse since then, and has had two attacks: in one unconscious, rigid, trembled and screamed; sleepy after it. Two attacks of aphonia. Left leg said to be drawn up at times. Very fat. Field of vision normal, *rod* 20-20, *ros* 20-100, astigmatism of 4 D, *os*. Quite tender over spine, and more or less tender all over. Smell, taste, and hearing normal. Slight diminution of electrical sensibility on left. Knee-jerks only on re-enforcement. In June, 1888, reported to have had chorea, and after that to have had an increase of all his symptoms, with one or two more attacks.

IX. Chas. D., 12, s., school-boy. Mother consulted me in September, 1886, for an opinion in relation to a suit for damages. Not neurotic. Two years ago he fell down a coal-hole, striking head. Unconscious for a time, and delirious for several days. Scalp-wound, and question of depressed fracture. Headache and vertigo since; could not go up or down stairs safely, owing to vertigo. Violent headaches induced by any effort or excitement. Very forgetful, peevish, fretful, and sleeps poorly. Has had attacks of severe headache, with nausea, and "raving attacks," when he would call out various phrases referring to his accident, and twisted about, his limbs working. Exhausted after these. Sudden attacks of pallor and vertigo. Some diplopia and tinnitus. Capricious appetite; has not grown much or gained in flesh since accident. Slight furrow over right parietal, and cicatrix over occiput. Some spinal tenderness in upper dorsal region; jarring causes vertigo. Field of vision and tactile sensibility normal. Special senses unimpaired. No knee-jerks. Gradual improvement, but in December, 1887, was still subject to severe headache and vertigo on any exertion or excitement. No attacks since May, 1886.

X. Charles C., 61, w., machinist. June, 1887. No special taint. Eighteen months ago he was thrown from a wagon, and was unconscious for six and a-half hours after it. No external injury or fracture. Hot water bottles were put to his feet, burning them so badly that he was kept in bed for four months. On getting about, his shoulders and right leg began to feel heavy and his arms ached. His head felt sore, and he has had sharp pains in it. Discouraged, low-spirited, and irritable, but mental power is not impaired. Short breath, poor appetite, constipation. Prickly aching and burning in the arms, which feel weak and heavy. Right leg feels numb and prickly, and both legs ache. He has pain and a hollow feeling in the back, which hinder his walking. Says he is growing worse. Marked myopic astigmatism, field of vision normal. Fibrillary tremor of tongue. It hurts him in the lower dorsal region to bend his spine, but it is not tender. Some inco-ordination

of the left arm, and a little tremor of the hands. Speech is a little thick. Epigastric reflex present only on left. Tri-ceps, radial, ulnar, and patellar reflexes exaggerated. Slight patellar clonus; front-tap contraction. March, 1888: Condition not improved. Complains greatly of his back, and of inability to use his arms well. Numbness of both legs. No inco-ordination of hand. Reflexes exaggerated. No consciousness of events immediately preceding accident. Was thrown from a carriage fifteen years ago, and after that had some stiffness of left arm, which recovered. A year before his last accident, however, this arm had been rather weak.

XI. Dennis B., 37. m., printer. Referred to me by Dr. Post, in August, 1885. In January, 1885, was struck by shafting, the right side being most injured. Right instep and right little finger broken; right thigh and leg much bruised. Laid up until May. Two weeks ago tried to go to work on a hot day, worked an hour and a-half, and had to go to bed. Memory began to fail after injury, and he has had constant severe headache ever since. Has vertigo so badly that his wife is afraid to let him go out alone. Forgetful since his injury. Very restless at night. Much more irritable and excitable. Considerable diplopia. Slight palpitation and shortness of breath. Poor appetite, some vomiting. At times has to wait before he can pass water, and at times the stream stops. Cannot close right hand as well. Frequent and severe pain in the right leg after using it, and constant numbness and prickling. Right leg weaker, somewhat wasted, and is easily fatigued. After his attempt to go to work, was in bed for a week; headache and vertigo much worse, felt dazed, and has been more or less confused; had constant nausea and vomiting for a week. Field of vision good, *rod* 20-20, *vos* 20-30, left disk paler. Some weakness of external recti, with nystagmus on excursion outwards. Other senses and tactile sensibility unimpaired. Arms strong, no inco-ordination, some tremor of hands. Right leg smaller than left, vastus internus does not react to faradism, lower leg muscles require a stronger current than on left. Nerve-trunks in right leg rather tender.

Reflexes normal. His symptoms gradually increased ; he grew steadily weaker, the mental deterioration became more marked, and about a year later he died. No autopsy.

XII. Wm. M., 49, m., engineer. Consulted me in March, 1887. Gonorrhœa. Considerable tobacco, little alcohol. Struck by a stone from a blast twenty years ago, breaking right forearm and thigh, and cutting radial (?) nerve in upper arm. Anchylosis of right elbow joint. Arm has been partly paralyzed since. Ever since accident has been nervous, "shattered." Sleep is very restless ; feels unstrung. Any excitement or any considerable exertion uses him up, and makes him put out of breath. Some "rheumatic" pain in legs. Cannot move right arm at shoulder much, or at elbow at all ; can flex and spread wrists and fingers, but cannot extend them ; supinates a little, pronates well. Arm two or three inches smaller round ; muscles wasted, do not react to faradism. Diminished sensibility in distribution of radial nerve. Sensation, motion, and reflexes elsewhere normal. March, 1888: Condition much the same. The pain in the legs is of rather recent date, and is of a rheumatic character. He is able to work, but since his injury he has been of a nervous, unstable disposition. Before it he could stand anything ; since, everything excites him very much, and makes him very nervous.

These last two cases are of further interest from the fact that Erichsen²² claims that when injury produces fracture of any bone, the nervous system is apt to escape from the effects of concussion, the violence of the shock expending itself in producing the fracture. These cases, as well as a good many others that have been reported, show at least that Erichsen's rule is not without exceptions.

These cases certainly present divers groups of symptoms which demand a little consideration. The commonest among them point to some cerebral disturbance. Eight had headache or pain in the head, and eight had vertigo ; ten had some psychical disturbance, nervousness, restle-

²² J. E. Erichsen. *Op. cit.*, p. 73.

ness, irritability, inability to make prolonged effort, depression, anxiety, loss of memory, and, in at least one case (XI.), distinct mental impairment; two (VIII. and IX.) had some sort of convulsive seizure; one only (IX.) seemed to have been affected by any terror, and in him the effect was slight.

Motor disturbances were not uncommon. Seven patients had muscular weakness, which sometimes amounted to actual paralysis, although chiefly when there was neuritis. Several had tremor; two (V. and VI.) Romberg's symptom; and one (X.) inco-ordination. Several had muscular wasting, and four diminished electrical excitability, chiefly from neuritis. The knee-jerks were increased in three cases, diminished in three, and absent in one.

Sensory disturbances were less common. In only four cases was there poor vision, due generally to definite causes independent of the injury. In three cases the other special senses were impaired, generally on one side. Three patients had diplopia, and one (XI.) nystagmus. One (VII.) had monocular diplopia, sluggish pupils, and xanthopsia; the last symptom was noted in one of Oppenheim's cases. Contracture of the field of vision was found but once, but in a few instances the fields were not examined. Anæsthesia in varying degrees was noted in seven cases, due, in two at least, to neuritis. In four of these seven cases, and in two others, there was paræsthesia. In no case, unless possibly in Case VIII. was the anæsthesia strictly unilateral—hemi-anæsthesia.

Digestive disturbances were occasionally seen, and in five cases there were vesical symptoms—signs of paresis of the bladder. Two men reported impotence.

Pain in the back was found in seven cases, and several others had pain in the side, limbs, or abdomen. The pain in the back was usually associated with tenderness over the spinal muscles and was increased on motion. In a few instances it was associated with tenderness over the spinous processes.

What is the cause of such an array of symptoms? Is there "only hysteria, nothing but hysteria?" Is there

merely a strain of the muscles of the back, with neurasthenia added to it? Is there merely a functional derangement, or is there some structural change in the nervous system? Of course it is not possible to find any one diagnosis to fit so many different cases, but these cases and their attendant symptoms may furnish us with some data to aid in considering the whole subject of so-called "concussion of the spine."

Before discussing the question further it is essential to give some sort of a definition of what is meant by the two vague terms hysteria and neurasthenia. Neither of them can rightly be regarded as a disease. They are both conditions of the individual, the latter being well defined as "a bodily condition which is frequently associated with various chronic disorders, and not rarely coexisting with perverted functional activity of the nervous centres."³³ These states are often erroneously spoken of as if they were distinct diseases, and the names are often used as convenient "dumps" for cases where we can make no diagnosis. Hysteria is the state in which ideas can control the body and produce morbid changes in its functions," while neurasthenia is a state of exhaustion from over-strain. The two states may be combined, or may complicate other affections. There is a hysterical symptom-complex that is so definite that it may fairly well be spoken of as a disease, and that is the group of symptoms that make up the *grande hystérie* of Charcot. To that alone I will apply the term hysteria; other conditions I will speak of as the hysterical or neurasthenic states.

That psychical disturbance can produce functional paralyses has been known for many years, and these paralyses have been discussed under many different names, such as "emotional paralysis,"³⁴ "paralysis dependent upon idea,"³⁵

³³ H. C. Wood. *Nervous Diseases and their Diagnosis*, p. 18.

³⁴ P. J. Möbius. *Unter den Begriff der Hysterie*. *Centralblatt f. Nervenheilkunde*, 1st. Feb., 1888.

³⁵ R. B. Todd. *Clinical Lectures on Paralysis*.

³⁶ J. Russell Reynolds. *Remarks on Paralysis and other Disorders of Motion and Sensation dependent on Idea*. *British Medical Journal*, 6th Nov., 1869.

or the "Schrecklähmung" of the Germans.³⁷ It was reserved for Charcot,³⁸ however, to give us the explanation of their origin. He has found that in certain hysterical patients at La Salpêtrière, who were easily hypnotizable, he could produce, by suggestion when hypnotized, paralyzes precisely similar to those seen in other patients as the result of an injury. He therefore suggests that the mental state occasioned by the nervous shock of the accident is similar to the somnambulistic stage of hypnotism—there is an "obnubilation of the *ego*." The idea of injury occurring in this state of nervous shock or obnubilation, acts as a traumatic suggestion, producing the same results as ordinary suggestion in a hypnotized patient. The patient develops his own idea and suggests it.

Charcot's brilliant reasoning proves beyond question the existence of a traumatic hysteria in his cases, but, after a careful study of these cases, and of others collected by Rendu,³⁹ Poupon,⁴⁰ Lyon,⁴¹ and Berbez,⁴² I am unable to trace any resemblance between them and the cases cited above, or the cases reported by German observers. Charcot's patients present either typical grand hysteria, with hemianæsthesia of the skin and organs of special sense, and with hystero-epileptic attacks with grand movements, passionate attitudes, and the *arc de cercle*; hysterical monoplegia; or hysterical articular neurosis. The characteristics of hysterical monoplegia are paralysis, with or without contracture; anæsthesia of the paralyzed limb, not following any nerve tracts but having a sharp line of demarcation and encircling the limb like a bracelet; little or no atrophy, and normal electrical reactions. The joint affection is that first described by Brodie, simulating severe organic

³⁷ E. Leyden. *Op. cit.*, i. 173.

³⁸ J. M. Charcot. *Op. cit.*, iii. 355, 392 *et seq.*

³⁹ H. Rendu. Contribution à l'histoire des monoplégies partielles du membre supérieur, d'origine hystero-traumatique. *Archives de Neurologie*, Sept., 1887.

⁴⁰ H. Poupon. Paralysies hystero-traumatiques. *L'Encéphale*, Jan., 1886.

⁴¹ G. Lyon. Note sur l'hystérie consécutive aux traumatismes graves. *L'Encéphale*, Jan., 1888.

⁴² P. Berbez. *Hystérie et Traumatisme*. Paris, 1887.

disease, and attended with great pain, cutaneous hyperæsthesia, and tenderness and stiffness of the joint, the stiffness disappearing under ether. The diagnosis of these conditions is not difficult, and in the cases reported above they were not present. I have seen all three of the conditions,—grand hysteria, hysterical monoplegia, and articular neurosis; but I have not yet seen these conditions appearing as the result of injury.

The researches of Thomsen and Oppenheim have shown conclusively that hemianæsthesia is not pathognomonic of hysteria. Charcot formerly held ⁴³ that general anæsthesia was rare in hysteria, that the anæsthesia was usually unilateral, the median line forming the limit. This claim has been abandoned, for many of his cases show anæsthesia of only one limb, and Berbez has found hemianæsthesia in only thirty-eight out of ninety-three cases. The commonest symptom in sensory anæsthesia—a point in which both French and Germans observers agree—is the peripheral limitation of the field of vision. Thomsen and Oppenheim have found sensory anæsthesia, which in many cases was unilateral, in epilepsy, hysteria, neurasthenia, nervousness, chorea, “railway-spine,” multiple sclerosis, Westphal’s pseudo-sclerosis, organic cerebral disease, certain psychoses, and other conditions; peripheral limitation of the visual field being the most constant symptom. Furthermore, the investigation of the committee of the Société de Biologie, consisting of Charcot, Luys, and Dumontpallier, have shown ⁴⁴ that in hemianæsthesia due to undoubted organic disease of the brain the phenomena of transfer can sometimes be excited by the application of metals. Thus it seems that not only is hemianæsthesia not pathognomonic of hysteria, but also that transfer phenomena in hemianæsthesia are not pathognomonic of hysteria.

Thomsen and Oppenheim oppose the hypothesis of hysteria as an explanation of their cases on other grounds. To their argument that the anæsthesia is fixed and un-

⁴³ J. M. Charcot. *Leçons sur les localisations*, etc., p. 115.

⁴⁴ Premier Rapport fait à la Société de Biologie sur la métalloscopie et la métallothérapie du Dr. Burq. *Gazette Médicale*, 28th April, 1877.

varying, and that the disposition is not that of the hysterical, Charcot has replied by citing cases where the anæsthesia in hysterical patients has not varied for years. Oppenheim's further objections, however, seems to be more conclusive. He finds that the patients are anxious, hypochondriacal, and depressed; complaining of headache, vertigo, faintness, and occasionally of epileptiform attacks. They have pain in the back, anæsthesia, and peripheral limitation of the field of vision; their movements are slow and feeble; and they try to guard their spines from jarring; there is often tremor; the vesical functions may be disturbed; the pulse is often rapid; in one case Oppenheim found unequal pupils which did not react to light, in another optic atrophy. This latter symptom is not very rare; it has been noted, for example, in cases reported by Rigler,⁴⁵ Walton,⁴⁶ and Wharton Jones.⁴⁷ Not only are these symptoms not hysterical, but they are not even functional; and Oppenheim justly argues that if they be ranked as hysterical the firmest pillars of our knowledge are overturned. Westphal,⁴⁸ too, asserts that these cases cannot be brought under the rubric of hysteria,

Within a few months Oppenheim has reviewed the subject again,⁴⁹ and concludes that the cases with signs of undoubted organic disease, immobile pupils, optic atrophy, and vesical symptoms are in the minority, and that most cases represent, not a typical neurosis like hysteria or neurasthenia, but a more general and complex psychoneurosis, from which the patient never recovers.

In the cases reported above the leading symptoms were certainly not those of hysteria. The psychical conditions

⁴⁵ J. Rigler. Ueber die Folgen der Verletzungen auf Eisenbahnen, insbesondere des Verletzungen des Rückenmarks. Berlin, 1879.

⁴⁶ G. L. Walton. Art. cit., Boston Medical and Surgical Journal, 11th Oct., 1883.

⁴⁷ Quoted by Eichhorst. Handbook of Practical Medicine. Am. Trans., iii. 144.

⁴⁸ Archiv f. Psychiatric u. Nervenkrankheiten, xvii. 282. 1886.

⁴⁹ H. Oppenheim. Wie sind die Erkrankungen des Nervensystems aufzufassen, welche sich nach Erschütterung des Rückenmarkes, insbesondere Eisenbahnunfällen, entwickeln? Berliner klinische Wochenschrift, No. 9, 27th Feb., 1888.

noted—depression, anxiety, loss of memory, mental impairment; the tremor; the exaggerated reflexes, and the swaying with closed eyes; the pronounced paræsthesia; the vertigo and headache (persistent headache being confessedly not a symptom of hysteria);⁵⁰ nystagmus; vesical paresis—all these point to something beside hysteria or the hysterical state. Moreover, incontinence of urine, nystagmus, and exaggerated reflexes are symptoms which we should expect to find in organic rather than in functional disease. Case XI., especially, is strongly suggestive of disseminated sclerosis, and the fatal termination renders the diagnosis of organic disease assured.

It seems to me that the theory of an organic lesion, possibly the lesions suggested by Westphal, is the one which is the most satisfactory in many of these cases. Bramwell,⁵¹ who is certainly sceptical as to the frequency of organic change, suggests that there may be multiple capillary hæmorrhages in the brain and cord, which give rise to inflammatory processes, and finally to sclerosis. These hæmorrhages are so small as easily to escape notice, and later, if there be sclerosis, the recent methods of staining would be necessary to detect it. An interesting corroboration of this theory is afforded by the general lesions found in the bodies of some of those killed at the great railway accident at Charenton in 1881. In several cases Vibert⁵² states that there were found very abundant punctuate hæmorrhages in the upper part of the body, and suggests that they arose from lesion of the nervous centres. Willigk⁵³ found in one case dilatation of the finest vessels, with infiltration into the perivascular spaces, and degeneration of the coats of the vessels. Page,⁵⁴ however, asserts that in these railway cases "Mors silet;" which is as correct as many of his statements. Autopsies and experimental pathology furnish us with various facts which prove an organic change.

⁵⁰ J. M. Charcot. *Leçons sur les maladies du système nerveux*, iii. 268.

⁵¹ B. Bramwell. *Diseases of the Spinal Cord*, 2d ed., p. 312.

⁵² Ch. Vibert. *Etude médico-légale sur les blessures produites par les accidents de chemin de fer*. Paris, 1888.

⁵³ A. Willigk. *Anatomischer Befund nach Hirnerschütterung*. *Vierteljahrsschrift f. die prakt. Heilkunde*, cxxviii. 19, 1875.

⁵⁴ H. W. Page. *Op. cit.*, p. 82.

In regard to experiment, Mendel,⁵⁵ believing that hyperæmia was an important feature of the early changes in general paralysis, sought to excite an intense chronic hyperæmia in dogs. For this purpose he fastened the animals on a revolving table, with their heads toward the periphery. Rapid revolution, 125 to 130 a minute, continued for half an hour, produced punctate hæmorrhages. Slower revolutions (110) for six minutes a day, produced, after some weeks, symptoms of general paralysis, and, on killing the animals, he found adhesions between the skull, the meninges, and the brain, an increase in the nuclei and cells of the glia, an increase in the number of vessels, and changes in the ganglion cells. This condition finds a clinical representation in a case recently reported by Bernhardt,⁵⁶ where symptoms of general paralysis developed gradually after a railway injury. Fürstner⁵⁷ has repeated Mendel's experiments, with fewer revolutions (60 to 80) for a shorter time (1 to 2 minutes) and continued for months. He found double primary degeneration of the lateral columns and of a particular part of the posterior columns, changes in the optic nerves and changes in the brain similar to those found by Mendel. Similar changes in the lateral columns have been found after death, in patients who had suffered from "concussion," by Dumenil and Petel, and also by Edes,⁵⁸ who has called attention to the occurrence of symptoms of spastic paralysis in certain cases.

In undoubted organic disease, however, no matter what the case may be, there are often symptoms which are due to a superinduced hysterical or neurasthenic state, and these symptoms may so overlay the symptoms due to the structural changes as to render the diagnosis extremely

⁵⁵ E. Mendel. Ueber paralytischen Blodsinn bei Hunden. Ref. in *Neurolog. Centralblatt*, 15th May, 1884.

⁵⁶ M. Bernhardt. Beitrag zur Frage von der Beurtheilung der nach heftigen Körpererschütterungen, in specie Eisenbahnunfällen, auftretenden nervösen Störungen. *Deutsche medicinische Wochenschrift*, 29th March, 1888.

⁵⁷ Quoted by Hoffman, art. cit.

⁵⁸ R. T. Eddes. The somewhat frequent occurrence of Degeneration of the Posterior-lateral Columns of the Spinal Cord in so-called Spinal Concussion. *Boston Medical and Surgical Journal*, 21st Sept., 1882.

difficult. I did not rehearse these symptoms ; they were present in several cases where I believe there was also organic disease, and I have seen organic disease not due to injury so masked by hysterical or neurasthenic symptoms as to make the case a puzzle.⁵⁹ One symptom of the neurasthenic state is not very rare, and that is the hyperæsthesia over the spinous processes of the vertebræ.

Apart from this mixture of functional and organic symptoms we must, however, recognize the fact that there are two great classes of cases which are the result of railway accidents and other injuries,⁶⁰ one where the symptoms are due, as I have endeavored to prove, to organic changes in the central nervous system ; the other where the symptoms are due to functional disturbance.

As a subdivision of this latter class may be mentioned the more purely psychical disturbances, which are not rare. Naturally these are seen more markedly after railway accidents than after the accidents of ordinary life, such as falls. The horror of a scene like that at Roslindale is not soon forgotten by the ordinary spectator, and to one who has been an active participant, with the terror of sudden death or some fearful injury imminent, the effect must be still greater. Immediately after the accident at Charenton Vibert⁶¹ noted a pronounced psychical change in almost all of the survivors, characterized by nervous excitability, insomnia, frightful dreams when sleep did come, tremor, headache, and a sort of semi-consciousness or cerebral automatism. This condition, he states, disappeared after a short time,—a few days or weeks,—but I believe that such disturbances are often of much longer duration ; at any rate I have known the psychical shock of a railway accident to be apparent for a good many months afterwards. Moeli⁶² has noted some of the more permanent psychical condi-

⁵⁹ For a fuller discussion of this point see E. C. Seguin's article on Hysterical symptoms in Organic Nervous Affections, in his *Opera Minora*, p. 180.

⁶⁰ F. Kalliefe. Ueber Rückenmarkerschütterung nach Eisenbahnunfällen (Railway-spine), p. 27.

⁶¹ C. Vibert. *Op. cit.*, pp. 11, 35.

⁶² C. Moeli. Ueber psychische Störungen nach Eisenbahnunfällen. Berliner klinische Wochenschrift, 7th Feb., 1881.

tions. The patient becomes sensitive to sensory impressions, and they are irritable, anxious, and depressed. They are easily fatigued, weak, nervous, tearful and tremulous; they are unstable and incapable of mental application, and all thought becomes difficult. They generally have headache, which is increased by mental effort or by slight amounts of alcohol. Moreover they are dominated by the thought of the accident, which is a constant source of oppression to them and haunts them night and day. Thomsen⁶³ has lately described a case under the name of "acute railway brain," where there was a slightly different symptom-complex. Immediately after the accident there were maniacal symptoms, with absolute and complete anæsthesia, confusion, and delusions of persecution. Latter the maniacal symptoms disappeared, the anæsthesia was less complete, but the man became lachrymose, hypochondriacal, depressed, irritable and unable to work on account of headache and weakness.

Beside these psychical disorders there are other manifestations of functional nervous disease. I believe that hysteria (the "grand hysteria" of Charcot) is only rarely the result of injury, but there are a large class of cases which, after injury, suffer from symptoms which are a part of the neurasthenic state. After severe concussion, or the psychical trauma of injury, the victim is thrown into a pronounced neurasthenic state, which may last for years. His nervous system is utterly shattered, or, to use the phrase of the day, he is "all broken up." He is nervous, emotional, irritable, perhaps hysterical; he is overcome by trifles; he is exhausted by the slightest effort. He may present no objective symptoms, but he remains an utter wreck. There is a general weakening, and a decline from the normal standard in the functions of the central nervous system, especially in the domain of the thought, the will, and the emotions.⁶⁴ The symptoms may be milder, as in Case XII. Here, the

⁶³ R. Thomsen. Vorstellung eines Falles von acutem schweren Railway Brain. Verhandl. der Gesellschaft der Charité-Aerzte in Berlin. Berliner klinische Wochenschrift, 18th Aug., 1887.

⁶⁴ M. Bernhardt. *Art. cit.*

patient finds himself changed : instead of being capable of continued exertion or strain, he is easily upset, trifles annoy him, he is irritable and quick tempered, he has lost the power of rebounding after pressure, of maintaining the calm, good tempered spirit which perhaps he had before ; his sleep is not sound, he starts when a door slams, his children annoy him, he is fretful and fault-finding. It may be that such a man is able to work as well as before, and to earn as much money, but, if he be in some responsible position, perhaps his judgment is less sure, or his bearing toward his associates less agreeable ; he is no longer a "good fellow," but nervous and disagreeable. These things, of course, are trifles, for which no jury would award damages ; but they are trifles which mark an enfeebled nervous system, and it is these very trifles which are like sand in the bearings of the carriage : they decide whether life is agreeable or disagreeable, and they are trifles which may continue for years ; in fact, the man may never recover his old buoyancy, his consideration for others, and his good nature.

I have not spoken as yet of the subject of strain of the muscles of the back, upon which so much stress has been laid by Page. It is, I believe, the chief source of the marked rigidity of the spine so often seen, and by it or by "spinal irritation" is to be explained the spinal tenderness so often met with, for spinal tenderness has as little to do with disease of the cord as pain in the back has to do with renal disease. Although, however, it is often present, I believe that it usually exists as a complication and not as the sole cause of the symptoms. The first two cases reported are good examples of muscular strain, but in neither of them was that the only trouble. The first man had paræsthesia in the arms, vertigo, psychical disturbances, and vesical symptoms, and the second had vesical disturbance and impotence. Page⁶⁵ seeks to ascribe incontinence and impotence to the strain alone, but I am unwilling to accept his conclusions. With strain of the muscles of the back there may be a little difficulty in emptying the bladder, but, when there is pain only on motion, I cannot see how

⁶⁵ H. W. Page. *Op. cit.*, p. 182.

the strain should paralyze the sphincters. In severe twists of the spine the nerves may be implicated, and it is possible that some of the nerves of the vesical plexus may be among them, yet that is by no means clear. Impotence is so common in all conditions of pain or weakness as to be of no significance. Strain of the muscles may often present such prominent symptoms as to mask the symptoms of nervous disturbance that lie beneath, yet I believe that careful inquiry will, in most cases of "concussion," reveal symptoms referable to the nervous system. As to Page's claim⁶⁰ that these cases do not recover owing to excessive doses of bromide of potassium, it is too much the argument of the railway attorney to merit consideration.

Of differential diagnosis I will say but little, because there is so much to say that it might easily fill a book; nor will I dwell upon the possibility of simulation, which has been so prominent in the minds of some writers as almost to conceal the fact that there was a real affection to be simulated. It is perhaps needless to add that all objective symptoms which indicate structural disease should be noted with care: the pupils and the optic nerve, the electrical reactions, the condition of the reflexes, the presence of tremor, especially fibrillary tremor of the face and tongue, ataxia, and Romberg's symptom. It should also be borne in mind that peripheral limitation of the field of vision, which is regarded by Thomsen and Oppenheim as one of the most constant attendants upon anæsthesia, and is considered by Charcot one of the chief stigmata of hysteria, may be the earliest recognizable manifestation of atrophy of the optic nerve.

In spite of the most rigid examination, however, there are many cases which furnish us no objective signs, notably the cases of purely psychical disturbance, and the conditions of neurasthenia, the purely "functional" affections. Here we can say only this, that if the patient's statements be true, he has suffered severe and perhaps incurable injury. If, in such cases, we had the opportunity of long-continued

⁶⁰ H. W. Page. On the Abuse of Bromide of Potassium in the Treatment of Traumatic Neurasthenia. *Medical Times and Gazette*, 4th April, 1885.

and constant observation, we would be in a better position to judge of the patient's veracity; but it may be as difficult to decide, and may require as long-continued study as it does in certain cases of insanity.

The prognosis in these cases varies, of course, with the character of the affection. Although meningeal hæmorrhage may be absorbed, other distinct structural affections of the brain or cord, of course, seldom recover. A noteworthy instance of recovery after paralysis of four years' duration is the case reported a number of years ago by Dr. Webber.⁶⁷ The prognosis of neuritis, which was present as a complication in several of the cases I have reported, depends, of course, upon the amount of injury to the nerve as shown by the electrical reactions. Strain of the muscles of the back, by itself, is an affair of long duration, and, as a complication, I have found it very persistent, lasting several years. Never having seen grand hysteria as a result of trauma, I am unable, from my own experience, to judge of the prognosis; but I see no reason why, when a person is once thrown into that state, from injury, he should recover, in the sense of getting out of the state, any quicker than one who gets into it from other causes, except that the absence of a hereditary nervous taint is in his favor. I suspect, however, that comparatively few cases of hysteria are induced from injury in persons who have not the taint. Charcot's cases are chiefly "héréditaires," but Berbez and Lyon are in doubt as to the preponderance of hereditary taint in the cases reported. Their conclusions are less decisive, however, as many persons, without heredity, may be of a neuropathic type. I find my own opinion supported by the recent testimony of Bataille: "It is apparent that predisposition is alone capable of making us comprehend the development of these attacks of hysteria." Of course, in hysterical cases the individual symptoms—anæsthesia, paralysis, contracture—may disappear, but the underlying state remains, capable of reproducing all the symptoms at short notice.

⁶⁷ S. G. Webber. Recovery after four years' paralysis following railroad injury. *Boston Medical and Surgical Journal*, 18th July, 1872.

What has been said of the hysterical state holds true in a measure of the neurasthenic state. Where the symptoms are mild, and there is no neurotic taint, the patient's chances of recovery are of course better. Even in severer forms of neurasthenia the prognosis is not utterly bad, although, unfortunately, there are many cases that never recover, whether the neurasthenia be of traumatic origin or not. Nevertheless, in many cases, even though there is so great improvement that the patient can return to work and do as much as before, there are still the subtler changes of which I have spoken which show that the recovery is not absolute.

The prognosis of purely psychical disturbances is also grave. Hartmann has also called attention to the fact that psychoses may develop a long time after an injury, especially if it has caused headache, vertigo, irritability, and a loss of power of intellectual application, as was seen in Case IX. Krafft-Ebing⁶⁸ also shows that injury may make the brain the place of least resistance; there is increased irritability, intolerance of heat or alcohol, and disturbance of vaso-motor innervation, which favors the development of psychoses. Thomsen also regards the prognosis of his "acute railway brain" as grave.

In the majority of cases the symptom-complex is something like this: the patient has headache and vertigo; he is depressed, irritable, and hypochondriacal, with a diminished power of application; he may have some visual disturbance, he often has a contracted field of vision and occasionally optic atrophy; there is some tremor and perhaps inco-ordination; he has some anæsthesia, usually not limited to one half of the body, and with it numbness and pricking; his movements are slow and weak; his tendon reflexes are exaggerated; there is often some lack of control over his bladder; and he may have pain and stiffness in the back from muscular strain. Here I believe the condition is due to a disseminated miliary sclerosis, or, in the early stages, to a hæmorrhage or inflammatory process. The prognosis is like that of multiple sclerosis. With rest, freedom from

⁶⁸ R. v. Krafft-Ebing. *Op. cit.*, i, 166.

excitement,—such as comes when litigation is over and the anxiety about money matters is settled,—and judicious treatment, the patient may improve. The same holds true of tabes dorsalis. The ultimate prognosis is bad. Oppenheim has never met with a recovery; Gowers⁶⁹ has found a good many cases where “damages” have not brought about a cure, and considers that where there has been a late or gradual onset of symptoms there is far less tendency to arrest or subsidence than with earlier lesions; and Strümpell,⁷⁰ in his latest edition, warns us against regarding these conditions as mild or insignificant. “The patients actually suffer greatly from them, and the suspicion of exaggeration and simulation is certainly much more rarely justified than it is pronounced.”

The following conclusions seem justifiable:

(1) Concussion of the spine, in the strict sense of the term, although probable, is still a matter of doubt.

(2) Muscular strain, spinal irritation, and peripheral neuritis are not uncommon complications.

(3) Injury may give rise, not only to gross mechanical lesions of the central nervous system, with symptoms coming on soon after the accident, but also to typical chronic degenerative processes of insidious onset.

(4) Injury may also give rise to various functional affections of the nervous system, including psychoses, hysteria, and neurasthenia.

(5) Hemianæsthesia is not pathognomic of hysteria, but is found in other conditions.

(6) Psychical disturbances—anxiety, hypochondriasis, depression, emotional disturbance, and lack of power of application—may exist alone or in conjunction with other affections.

(7) The neurasthenic state is often produced by injury, but true hysteria is rare.

(8) Both the hysterical and the neurasthenic states may be superimposed upon organic disease, obscuring the diagnosis.

⁶⁹ W. R. Gowers. *Op. cit.*, i. 453.

⁷⁰ A. Strümpell. *Op. cit.*, ii. 1, 164.

(9) There is a fairly typical symptom-complex, with psychical disturbances, paræsthesia, anæsthesia, slow and feeble movements, exaggerated reflexes, etc., which is not uncommon, and is probably due to organic disease.

(10) The prognosis of these conditions is grave. Improvement is not uncommon, but complete recovery is rare.

DISCUSSION ON DR. KNAPP'S PAPER.

Dr. SEGUIN referred to the difficulty of diagnosis. As elements of error he mentioned uncertainty or unconscious deception by the patient who ascribed to his injury symptoms which had commenced before the accident. To these were to be added intentional deception and auto-suggestion. The more enlightened school of hypnotism has demonstrated the production of various symptoms of nerve disease in the healthy person. Bernheim and his pupils have shown that these symptoms might be produced not only by the suggestion of the operator, but by the suggestion of the patient himself, in whom a firm belief and a constant thinking may result as in the hypnotic state in an apparent loss of power, anæsthesia, spasm, etc.

In regard to the hysterical element, he referred to a paper written in 1875, in which he had shown the frequent coexistence of hysterical symptoms with organic disease of the brain and cord. This combination was not peculiar to traumatism, but was found also in morphinism and after moral shock.

Dr. L. C. GRAY also alluded to the influence of suggestion. The psychical lesion was so large as to make the majority of cases undiagnosible. So soon as a person was injured he was besieged by runners for legal firms. By means of these runners and the lawyers he was impressed with the danger to which he had been subjected. The suit then followed, running on for two or three years, during which time the patient could not afford to get well, as he would thus become liable to a suit for conspiracy. Finally, after having kept up the disease for the two or three or four years during the long trial, habit would prolong it for at least three or four years after.

In several cases he had advised the counsel to settle the case, and recovery had occurred in a few months.

It was a curious fact that patients suffering from organic injury usually did not have neurasthenia. The exaggerated manner of the neurasthenic contrasted curiously with the calmness of the man with serious organic injury.

The psychical results of injury would entitle the sufferer to some recompense, but of course not to so much as military hemorrhages or other wide-spread disease.

Dr. ZENNER referred to the fact of hysterical symptoms conjoined with organic disease, referring to a case of lead poisoning associated with tremor, which disappeared under hypnotism and after two or three applications was cured. Deception on the part of the patient should not, on the other hand, lead the exclusion of other serious disease. Pure maligning was a very rare occurrence. Deception in itself presupposed disease.

Dr. KNAPP recognized the tendency to auto-suggestion and the exaggeration of symptoms produced by these medico-legal trials, but neither sources of error were present in his cases, as suits for damages were not in progress, and all had been anxious to get well.

The following paper was read :

THE CORTICAL LOCALIZATION OF THE CUTANEOUS SENSATIONS.

By CHARLES L. DANA, A.M., M.D., New York.

I desire to present the evidence, so far as it is now attainable, regarding the question of the cortical localization of the cutaneous senses, viz., tactile, thermic and pathic.

It is well known there are now two very antagonistic views with regard to this subject. Ferrier contends that these centres are in the hippocampal gyrus and gyrus fornicatus. He bases this view upon experiments upon eleven monkeys, certain anatomical considerations, and the clinical fact that very often lesions of the convex motor cortex of the brain do not cause any sensory symptoms. His ex-